Arteriovenous malformation of the jejunum

To the editor: Three years ago we treated a patient with an arteriovenous malformation as described in the following case report.

A 73-year-old man was admitted to hospital with weakness, dizziness and a decrease in hemoglobin concentration that had been detected in his family physician's office over the previous 3 weeks. He had no melena or change in stool colour, and no pain, nausea, vomiting or other symptoms.

The patient had a 25-year history of painless intermittent melena and anemia requiring transfusion "about 50 times". His only previous operations were two hemorrhoidectomies — one 20 years ago and another 4 years ago. The second had been preceded by full work-up for gastrointestinal tract bleeding. In recent years, on numerous admissions to several local hospitals, repeated investigation had been done by gastroenterologists and hematologists. No clues were found to the cause of his low hemoglobin concentration other than iron-deficiency anemia, and no therapy other than multiple transfusions and iron pills was given. His hemoglobin concentration would decrease to as low as 5.2 g/dL.

He appeared well developed and well nourished and was in no distress. The findings on physical examination, which included rectal examination, were normal; blood pressure was 150/75 mm Hg; pulse rate, 100 beats/min with a regular rhythm; and respiratory rate, 24/min. Hemoglobin concentration was 8.6 g/dL and hematocrit, 28%.

Since numerous barium radiographic studies and endoscopies had failed to disclose the diagnosis, selective superior mesenteric arteriography was performed. A dilated third jejunal artery — a branch of the superior mesenteric artery — led into multiple irregular vascular channels within the bowel wall; at this point early filling of a large jejunal vein was seen at the same level, extending over an 8- to 9-cm segment of jejunum.

At laparotomy a 70-cm length of the wall of the jejunum was found to be thickened and to contain large pulsating vessels. This portion was resected and an end-to-end anastomosis performed 10 cm distal to the ligament of Treitz. Pathologic examination revealed a thickened, congested jejunal wall with large arteries and veins in the submucosa but without edema or inflammation; no specific microanatomic vascular lesion was seen.

The patient's recovery was uneventful. In the 3 years since operation there has been no recurrence of anemia or bleeding.

The source of massive alimentary tract hemorrhage is usually confirmed by conventional methods such as barium studies and endoscopy. However, many patients with bleeding from the upper gastrointestinal tract will be discharged from hospital without a diagnosis. In a series of 1000 cases analysed

by Palmer[1] the cause of the bleeding could not be discovered in 8%.

Since the introduction of angiography successful diagnosis of arteriovenous malformation of the bowel has been reported from time to time. Most of the malformations have been in the cecum and ascending colon, being fed by either the ileocolic or the right colic artery. In the Dartmouth study,[2] in which angiography was used in 22 patients with intestinal hemorrhage undiagnosed by barium contrast radiography, endoscopy or laparotomy, four such malformations were found, all in the cecum.

Of the 17 reported cases of angiographically diagnosed arteriovenous malformation in the bowel only 2 had jejunal malformations. The first was reported in 1968 in a 46-year-old woman with an 18-year history of intermittent melena.[3] In 1970 a 65-year-old man with a 10-year history of melena was treated at the Mayo Clinic; the jejunal arteriovenous malformation was angiographically demonstrated.[4] Both patients had had laparotomies without relief. Their histories were similar to that of our patient and seemed to be typical of the way in which these cases present.

Angiography is therefore an important diagnostic aid in the investigation of obscure bleeding from the gastrointestinal tract.

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Prostaglandins and the ductus arteriosus

To the editor: The editorial by E.M. Cooperman (*Can Med Assoc J* 117: 309, 1977) has drawn attention to the exciting new developments in our understanding of the role of prostaglandins in the regulation of ductus arteriosus closure. Yet, as so often happens, credit was not given to the Canadian investigators who have undertaken the work in this field. The work of Olley, Coceani and Bodach[1] was cited, but nowhere in the article could someone unaware of the story discover that the team that did much of the basic research and pioneered the clinical use of prostaglandins in children is based in Toronto. Surely this information ought to be of interest to readers of the Journal.

Cooperman failed to point out that, as a result of the Toronto group's findings of the importance of prostaglandins in maintaining patency of the ductus, two different approaches to the problem of the patent ductus were simultaneously and independently developed. The approach Cooperman mentioned — inhibition of prostaglandin synthesis by indomethacin and acetylsalicylic acid — was tried in the United States. In Canada it was found, on the basis of laboratory research, that the antimalarial chloroquine was an antagonist of prostaglandin action rather than an inhibitor of prostaglandin synthesis.[2] Because chloroquine has a record of safe use in neonatal malaria, Collins and colleagues,[3] at the Montreal Children's Hospital, tested it in infants with the patent ductus syndrome and obtained encouraging results. The use of indomethacin obviously causes problems and I believe that chloroquine may prove to be the better drug. The use of chloroquine was reported by me and my colleagues in your sister weekly journals — the *Journal of the American Medical Association*[4] and the *Lancet*.[5] It is unfortunate that the *CMA Journal* should pay more attention to the work being carried out in the United States than that being carried out in Toronto and Montreal.

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Chronic active hepatitis in a child with hemophilia A

To the editor: Hepatitis is a major complication of blood transfusion therapy, and the use of fractions prepared from large plasma pools has increased the risk. Both Factor VIII and Factor IX concentrates can be implicated as possible sources of the virus.[1-5] In view of this, one would expect to find antibody